

AD-A062 935

ARMY RESEARCH INST OF ENVIRONMENTAL MEDICINE NATICK MASS F/G 6/5
OBSERVATIONS ON ESOPHAGEAL TEMPERATURE DURING EXERCISE IN ASTHM--ETC(U)
AUG 78 E C DEAL, E R MCFADDEN, R H INGRAM
USARIEM-M-38/78.

UNCLASSIFIED

NL

1 OF 1
AD
AO 62935



END
DATE
FILMED

3 --79

DDC

Unclassified

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

REPORT DOCUMENTATION PAGE

READ INSTRUCTIONS
BEFORE COMPLETING FORM

1. REPORT NUMBER M 38/78	2. GOVT ACCESSION NO.	3. RECIPIENT'S CATALOG NUMBER
4. TITLE (and Subtitle) Observations on Esophageal Temperature During Exercise in Asthmatic and Non-Asthmatic Subjects		5. TYPE OF REPORT & PERIOD COVERED
7. AUTHOR(s) E. Chandler/Deal, Jr., E.R./McFadden, Jr., R.H./Ingram, Jr. James J./Jaeger		8. CONTRACT OR GRANT NUMBER(s)
9. PERFORMING ORGANIZATION NAME AND ADDRESS Departments of Medicine of Peter Bent Brigham Hospital & Harvard Medical School, Boston, MA 02115 & US Army Rsch Inst of Env Med, Natick, MA 01760		10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS
11. CONTROLLING OFFICE NAME AND ADDRESS (14) USARLEM-M-38/78		12. REPORT DATE (11) 8 August 1978
14. MONITORING AGENCY NAME & ADDRESS (if different from Controlling Office) S/A LEVEL II		13. NUMBER OF PAGES 1226p.
15. SECURITY CLASS. (of this report)		15a. DECLASSIFICATION/DOWNGRADING SCHEDULE
16. DISTRIBUTION STATEMENT (of this Report) S/A This document has been approved for public release and sale; its distribution is unlimited.		
17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report) Distribution of this document is unlimited.		
18. SUPPLEMENTARY NOTES		
19. KEY WORDS (Continue on reverse side if necessary and identify by block number) airway obstruction heat & water exchange in airways esophageal temperatures exercise 79 01 05 030		
20. ABSTRACT (Continue on reverse side if necessary and identify by block number) We measured upper esophageal temperature during exercise and the subsequent pulmonary functional responses in eight asthmatic subjects and in six normal subjects who breathed air at cold and dry, ambient and body conditions. Asthmatic subjects developed the greatest obstruction following exercise with cold and dry air and no response to air at body conditions, while the normal subjects developed no obstruction in the post-exercise period with any of the three air conditions. Despite the divergent responses in the two groups,		

DD FORM 1 JAN 73 1473

EDITION OF 1 NOV 65 IS OBSOLETE

040 850 Unclassified LB
SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

UUG FILE COPY AD A062935

upper esophageal temperatures fell by the same magnitude below core values in both normal and asthmatic subjects, indicating that identical degrees of airway cooling occurred. We conclude that rather than having a defect in the ability to condition inspired air, asthmatic subjects are more responsive to the effects of incompletely conditioned air.

ACCESSION NO.	
NTIS	<input checked="" type="checkbox"/>
DDC	<input type="checkbox"/>
UNANNOUNCED	<input type="checkbox"/>
JUSTIFICATION	
BY	
DISTRIBUTION	
Dist.	
A	

OBSERVATIONS ON ESOPHAGEAL TEMPERATURE DURING EXERCISE
IN ASTHMATIC AND NON-ASTHMATIC SUBJECTS

Running Title: ESOPHAGEAL TEMPERATURE AND EXERCISE-INDUCED ASTHMA

E. Chandler Deal, Jr., E. R. McFadden, Jr., R. H. Ingram, Jr. and James J. Jaeger
with the technical assistance of David Stearns and Sheryl Blake

From the Departments of Medicine of Peter Bent Brigham Hospital and Harvard
Medical School, Boston, MA 02115 and the U.S. Army Research Institute of
Environmental Medicine, Natick, MA 01760

Supported in part by Research Career Development Award HL-00013 (E. R. McF),
SCOR HL-19170 and Grants HL-17873, HL-17382, HL-16463 and HL-07010 from the
National Heart, Lung and Blood Institute

The views of the authors do not purport to reflect the positions of the
Department of the Army or the Department of Defense

Address correspondence to:
E. R. McFadden, Jr., M.D.
Pulmonary Disease Division
Peter Bent Brigham Hospital
Boston, MA 02115

79 01 05 030

- A -

ABSTRACT

We measured upper esophageal temperature during exercise and the subsequent pulmonary functional responses in eight asthmatic subjects and in six normal subjects who breathed air at cold and dry, ambient and body conditions. Asthmatic subjects developed the greatest obstruction following exercise with cold and dry air and no response to air at body conditions, while the normal subjects developed no obstruction in the post-exercise period with any of the three air conditions. Despite the divergent responses in the two groups, upper esophageal temperatures fell by the same magnitude below core values in both normal and asthmatic subjects, indicating that identical degrees of airway cooling occurred. We conclude that rather than having a defect in the ability to condition inspired air, asthmatic subjects are more responsive to the effects of incompletely conditioned air.

airway obstruction, heat and water exchange in airways, esophageal temperatures, exercise

INTRODUCTION

Recent reports, from this and other laboratories, have postulated that cooling of intrathoracic airways may be part of the initial reaction sequence by which exercise leads to airway obstruction in asthmatic individuals (1,3, 5-7,12). We have reasoned that if this were so, then one should be able to obtain some measure of the magnitude to which this occurs, as well as determine whether there are any differences between normal subjects who do not develop abnormal mechanics following exercise, and asthmatic subjects in whom bronchoconstriction occurs in direct proportion to the thermal burden placed upon their airways (6,7,12). Since direct recordings of the temperature in the tracheobronchial tree are technically difficult to obtain, particularly during exercise, we used the temperature in the esophagus adjacent to the trachea at the level of the carina as an indirect index of the changes taking place in the airways. Our observations form the bases of this report.

METHODS

Eight asymptomatic, asthmatic individuals with reproducible exercise-induced asthma previously documented in our laboratory, and six normal volunteers served as our subjects. Their anthropometric data are contained in Table 1. The asthmatics refrained from taking any medication for 12 hours before any study day, and none had used cromolyn sodium or glucocorticoids for at least one month before entering this investigation. Informed consent was obtained from each participant.

We defined the anatomic relationships of each subject's esophagus to other mediastinal structures by having them swallow a balloon-tipped catheter into the stomach via the nose. Pressures in the balloon were measured with a variable inductance transducer (Validyne MP-45-1) and recorded on a multichannel

time-based recorder (Hewlett Packard 7700-D). The catheter was slowly withdrawn so as to locate the gastroesophageal junction, maximum cardiac artifact and the lowest point in the esophagus at which movement of the trachea produced a pressure artifact (10). These distances were recorded and used to select the sites for the placement of thermal probes.

After the catheter was removed, the subjects then had two copper constantan thermocouples (Barly Instrument Co.) inserted through the nares into the esophagus. One was positioned behind the heart at the site of maximum cardiac artifact and the other at the point of the tracheal artifact. A third thermocouple was inserted approximately 10 cm into the rectum and used to measure core temperature. To insure that the esophageal probes maintained their relationships one to another, they were taped together before insertion, and then secured at the opening of the nares to avoid their movement within the esophagus during exercise. Each of the three thermocouples of a set were matched for response times and absolute readings in various thermal baths, spanning a 20 to 50°C range. The outputs from the thermocouples were recorded continuously and simultaneously during each experiment by a Neumatron Temperature Scanner (Leeds-Northrup) that was in series with a high speed teletype (Dec Writer Digital Equipment Corp.). This instrument complex digitized the analog data and gave a numeric temperature value from each thermocouple every seven seconds. The entire system was calibrated against a Bureau of Standards Thermometer immersed in water at various temperatures.

We measured airway resistance and total lung capacity with its subdivisions in a variable pressure plethysmograph that was serially interfaced to an analog recorder (Electronics for Medicine) and a minicomputer (Lab 8E, Digital Equipment Corp.) (8,9,15). Resistance was converted to its reciprocal, conductance, and expressed as a conductance volume ratio termed specific conductance (SG_{aw}) (2).

Four to five measurements of each variable were obtained, and the mean was computed. These data were considered acceptable if their coefficients of variation were 5% or less. Maximum forced exhalations were then performed in triplicate using a waterless spirometer (Electro Med. Model 780, Searle Cardio-pulmonary). 1-s forced expiratory volumes (FEV_1) and maximum mid-expiratory flow rates (MMF) were computed by standard techniques. The best effort, as defined by the curve with the largest forced vital capacity and FEV_1 , was used for analysis.

The temperature and water content of the inspired air was controlled by having the subjects breathe through a heat exchanger and bubble humidifier as in previous experiments (12). This set of instruments was capable of producing temperatures between -20°C and 120°C with relative humidities (RH) varying from zero to 100%. The temperatures of the inspired air were continuously recorded by a thermocouple located within the airstream in the exchanger, 10 cm upstream from the mouth. The output of this thermocouple was also fed into the temperature scanner and recorded at seven second intervals synchronously with the others. Expired gas was directed away from the exchanger through a one-way valve into a Tissot spirometer so that tidal volume and minute ventilation (\dot{V}_E) could be recorded. Heart rate (HR) was monitored continuously. The water content of the air supplied to the subjects was verified by drawing known volumes of air through glass drying tubes containing anhydrous calcium sulfate (W. A. Hammond Dricrite Co.) as previously described (12).

Esophageal and rectal temperatures were recorded before, during and after the subjects performed multiple bouts of exhausting leg work on a cycle ergometer while breathing air at various temperatures and water contents. In the asthmatics, air at ambient room, body and subfreezing conditions was employed in a random fashion; and in the normal subjects, room and subfreezing conditions

were used. In each of the experiments, the air was inhaled for four minutes before, during and for four minutes after the exercise period. Pulmonary mechanics were measured before and 5 to 10 minutes after cessation of work. The work loads, RPM and duration of exercise were held constant for each individual for each study. Upon completion of a work load, each subject rested for at least one and one half hours before subsequent exercise was undertaken. This time period was employed to allow pulmonary mechanics to return to pre-exercise levels if they had changed. These protocols exactly match those that we have routinely employed for exercise provocation of asthma in other studies (5-7, 11-14).

The data were analyzed by paired and unpaired t tests and one and two factor analyses of variance.

RESULTS

Table 1 contains individual data for anthropometrics, work loads, duration of exercise and the location of the esophageal thermocouples for the normal and asthmatic subjects. As a group, the normal subjects were older and larger than the asthmatics. The mean work loads were 956 ± 277 (SD) and 1250 ± 225 KPM for the asthmatics and normals, respectively. The mean duration of exercise was 3.9 ± 1.2 minutes in the asthmatics and 4.5 ± 1.4 minutes in the normals. There were no significant between group differences for these two variables.

The inspired air temperatures (T_i) and water contents (W_i) and the ventilatory and circulatory consequences of exercise are shown in Table 2. In the asthmatics, T_i and W_i were $-16.3 \pm 3.0^\circ\text{C}$ and 1.4 ± 0.3 mg $\text{H}_2\text{O}/\text{L}$ air; $26.8 \pm 1.0^\circ\text{C}$ and 6.2 ± 2.3 mg $\text{H}_2\text{O}/\text{L}$ air; and $37.2 \pm 0.5^\circ\text{C}$ and 44.7 mg $\text{H}_2\text{O}/\text{L}$ air in the cold, room temperature and body condition studies, respectively. In these experiments, the mean \dot{V}_E ranged from 68.8 to 70.4 L/min and mean HR from 161 to 163 beats/minute. The inspired air conditions used in the studies

involving normal subjects were statistically identical to those in the asthmatics, as were the heart rates with exercise. However, the \dot{V}_E achieved by the latter group was significantly higher. Expressing \dot{V}_E as a function of body surface area for both populations abolished this difference (e.g., \dot{V}_E/BSA asthmatics cold study = 45.3 ± 8.7 ; normal cold study = 49.2 ± 8.7 L/min/M²; $t = 0.92$; p NS). There were no significant differences for \dot{V}_E or HR between experiments for either group by one factor analysis of variance.

The effects of exercise on pulmonary mechanics for each inspired air condition are summarized in Figures 1 through 3. The baseline data for each variable for each experiment were identical. When the asthmatics inhaled cold air during exercise, SG_{aw} fell $55.2 \pm 16.6\%$ from its control value (Figure 1). Increasing the inspired air temperature and water content to 26.8°C and 6.2 mg H₂O/L air, respectively, (i.e., ambient room conditions) significantly altered the response. The fall from control was still significant (0.13 to 0.09 L/sec/cm H₂O/L), but the magnitude of the response was blunted in that the decrease in SG_{aw} was now only $43.0 \pm 9.3\%$. When air at body conditions was inhaled, no post-exercise change in mechanics developed. In the normal subjects, there was no change in SG_{aw} with exercise irrespective of the inspired air temperature. Similar patterns were observed for FEV₁ (Figure 2) and RV (Figure 3). These effects of T_i and W_i on the post exercise mechanical responses in asthmatics are quite similar to those previously reported (5-8,11,12).

Temperature recordings from the rectum and the two esophageal sites are shown for the asthmatics and normals in Figures 4 and 5. These data demonstrate that there were systematic differences in the temperatures of the three sites at rest, with the order being rectal greater than retrocardiac, which in turn was greater than retrotracheal. On the average, rectal temperature exceeds that in the esophagus behind the heart by 0.3°C, and that behind the lower end

of the trachea by 1.0°C in both groups of subjects. However, the important point is that with exercise, rectal and retrocardiac measurements kept a constant relationship one to another, and either did not change or rose slightly, while retrotracheal temperature fell significantly in the cold and ambient room experiments in both asthmatics (Figure 4) and normals (Figure 5). In the study in which air at body conditions was inhaled, retrotracheal temperature rose in concert with that in the retrocardiac position (Figure 4).

Comparison of the temperature differences between the retrocardiac and retrotracheal measurements for asthmatics and normal subjects during the last minute of exercise for the subfreezing and room condition experiments are shown in Figure 6. These data make several points. As can be seen, the lower the value for T_i , the greater the difference in temperature between the two esophageal sites (asthmatics, cold vs. room; $p < 0.001$; normals, cold vs. room, $p < 0.001$). However, for any given condition of T_i and W_i , there were no significant differences in the degree of cooling in either absolute or relative terms between populations.

Analysis of the association between the changes in retrotracheal temperature and pulmonary mechanics that developed with exercise under each inspired air condition in the asthmatics is displayed in Figure 7. One second forced expiratory volumes were chosen as a representative variable. The same relationship exists for SG_{aw} and RV. In this figure, it can be seen that the greater the fall in the esophageal temperature in the region of the trachea during exercise, the greater the degree of obstruction that developed. No such effect could be demonstrated in normal subjects despite an equivalent degree of cooling.

DISCUSSION

In previous studies we have shown that the degree of bronchial obstruction that develops following an exercise challenge in asthmatics is in direct proportion to the total respiratory heat exchange that occurs during exercise (6,7,12). In these experiments, airway constriction did not occur when the thermal burden placed upon the airways was eliminated by breathing air at body conditions and was most severe when subfreezing air was inhaled. Values for T_i and W_i between these two extremes resulted in intermediate degrees of bronchial obstruction. Because vagal efferent blockade did not prevent the potentiation of exercise-induced asthma produced by large thermal burdens (5), we postulated a direct effect of incompletely conditioned air.

In order for this postulate to stand, it was necessary to further hypothesize that incompletely conditioned air actually reached the intrathoracic airways. If this occurred, these airways would have to give up heat and water from their mucosa in order to bring the inspire to body conditions. They, in turn, should be cooled with the temperature drop being in direct proportion to \dot{V}_E , and in indirect proportion to the water content and temperature of the inspire. The results of the present study clearly demonstrate that the temperature of the intrathoracic airways does indeed fall with magnitude in indirect proportion to T_i and W_i . Further, in the asthmatics there is a clear relationship between the degree of obstruction and the absolute fall in retro-tracheal temperature during exercise (Figure 7). Since the same temperature changes occurred in normals in whom airway obstruction did not develop, we believe it unlikely that asthmatics have a defect in the way they condition inspired air. Rather, it appears that the asthmatic response is another manifestation of their increased airway reactivity.

Essential to our interpretations is that our upper esophageal measurements are a reasonable index of the temperature in the adjacent airways. Quite probably the values are considerably higher than would be found in the airway mucosa and certainly higher than in the airstream, since tissue perfused by blood at core temperature intervenes between the esophageal site of measurement and the structures whose cooling we have postulated should occur. Thus, if anything, we are underestimating the magnitude of airway changes. The fact that lower esophageal temperature closely matched both in sign and magnitude the change seen in the rectal probe would rule out a spurious result due to swallowing of air or cooled saliva, since both upper and lower esophageal probes registered identical, simultaneous temperature decreases when either cold air or water was deliberately swallowed. Hence, the direct relationship between upper esophageal temperature drop and the magnitude of both the thermal burden and the degree of post exertional obstruction that developed in the asthmatics argues strongly that, despite the thermal buffer of perfused tissue, we were assessing a reasonable index of temperature in the airways.

Similar conclusions were reached by Cranston and colleagues in a study concerned with the factors affecting oral, rectal and esophageal temperatures in normal man (4). In this investigation, esophageal temperature was observed to fall progressively from cardia to mouth with the chief change occurring at depths of 30 cm from the nares. (This position corresponds quite favorably to the 30.6 and 32.0 cm locations of the retrotracheal probes in our two groups of subjects). The cause of this behavior was believed to be due to the close apposition of the trachea and esophagus in the upper thorax.

In summary, we have confirmed that the magnitude of post-exertional airway obstruction in asthmatic subjects is a function of the thermal burden during the hyperpnea of exercise and have shown that the temperature drop in the

airways, as assessed by upper esophageal temperature, relates directly to both. In contrast to asthmatic subjects, normal subjects developed no post-exertional obstruction despite similar degrees of airway cooling. Thus asthmatics, rather than having a defect in conditioning of inspired air, would appear to be more responsive to the effects of incomplete conditioning.

REFERENCES

1. Bar-Or, O., I. Neuman, and R. Dotan. Effects of dry and humid climates on exercise-induced asthma in children and pre-adolescents. *J. Allergy Clin. Immun.* 60:163-168, 1977.
2. Briscoe, W. A., and A. B. DuBois. The relationship between airway resistance, airway conductance, and lung volume in subjects of different age and body size. *J. Clin. Invest.* 37:1279-1285, 1958.
3. Chen, W. Y., and D. J. Horton. Heat and water loss from the airways and exercise-induced asthma. *Respiration* 34:305-313, 1977.
4. Cranston, W. I., J. Gerbrandy, and E. S. Snell. Oral, rectal and esophageal temperatures and some factors affecting them in man. *J. Physiol.* 126:347-358, 1954.
5. Deal, E. C., Jr., E. R. McFadden, Jr., R. H. Ingram, Jr., and J. J. Jaeger. Effects of atropine on the potentiation of exercise-induced bronchospasm by cold air. *J. Appl. Physiol.: Resp. Envir. Exercise Physiol.* 45:238-243, 1978.
6. Deal, E. C., Jr., E. R. McFadden, Jr., R. H. Ingram, Jr., R. H. Strauss, and J. J. Jaeger. The role of respiratory heat exchange in the production of exercise-induced asthma. *J. Appl. Physiol.: Resp. Envir. Exercise Physiol.* (Submitted for publication).
7. Deal, E. C., Jr., E. R. McFadden, Jr., R. H. Ingram, Jr., and J. J. Jaeger. Hyperpnea and heat flux. The initial reaction sequence in exercise-induced asthma. *J. Appl. Physiol.: Resp. Envir. Exercise Physiol.* (Submitted for publication).
8. DuBois, A. B., S. Y. Botelho, G. N. Bedell, R. Marshall, and J. H. Comroe, Jr. A rapid plethysmographic method for measuring thoracic gas volume: A comparison with a nitrogen washout method for measuring functional residual capacity in normal subjects. *J. Clin. Invest.* 35:322-326, 1956.

9. DuBois, A. B., S. Y. Botelho, and J. H. Comroe, Jr. A new method for measuring airway resistance in man using a body plethysmograph: values in normal subjects and in patients with respiratory disease. *J. Clin. Invest.* 35:327-335, 1956.
10. Milic-Emili, J., J. Mead, J. M. Turner, and E. M. Glauser. Improved technique for estimating pleural pressure from esophageal balloons. *J. Appl. Physiol.* 19:207-211, 1967.
11. Strauss, R. H., E. R. McFadden, Jr., R. H. Ingram, Jr., and J. J. Jaeger. Enhancement of exercise-induced asthma by cold air breathing. *N. Eng. J. Med.* 297:743-747, 1977.
12. Strauss, R. H., E. R. McFadden, Jr., R. H. Ingram, Jr., E. C. Deal, Jr., and J. J. Jaeger. Influence of heat and humidity on the airway obstruction induced by exercise in asthma. *J. Clin. Invest.* 61:433-440, 1978.
13. Strauss, R. H., R. H. Ingram, Jr., and E. R. McFadden, Jr. A critical assessment of the roles of circulating hydrogen ion and lactate in the production of exercise-induced asthma. *J. Clin. Invest.* 60:658-664, 1977.
14. Strauss, R. H., R. L. Haynes, R. H. Ingram, Jr., and E. R. McFadden, Jr. Comparison of arm versus leg work in the induction of acute episodes of asthma. *J. Appl. Physiol.: Resp. Envir. Exercise Physiol.* 42:565-570, 1977.
15. Sykes, T. W., R. L. Haynes, and E. R. McFadden, Jr. On line determination of lung volumes by plethysmography and digital computer. *Am. Rev. Resp. Dis.* 115:581-585, 1977.

TABLE 1

Anthropometric data, work loads, duration of exercise and thermocouple distances

ASTHMATICS

Subject	Age	HT	WT	Work Load	Duration	Location of Thermocouples Retrotracheal	Retrocardiac
TW	27	170.2	65.9	1200	4.4	32	47
FF	24	177.8	76.4	1200	6.2	32	47
LK	22	160.0	54.6	750	4.0	30	45
ML	21	157.5	54.6	600	4.3	29	39
PM	24	160.0	68.2	900	4.5	29	39
MS	24	157.5	50.0	600	2.7	29	39
DM	24	183.9	68.2	1200	2.6	32	45
MR	19	177.8	68.2	1200	2.9	33	46
MEAN	23.1	168.1	63.3	956	3.9	30.6	43.4
SD	2.4	10.7	9.1	277	1.2	1.5	3.7

NORMALS

ND	26	185.4	71.4	1200	4.3	34	47
CO	30	185.4	86.4	1500	6.2	33	46
NS	31	180.3	75.5	1200	3.5	32	45
SP	31	182.9	75.5	1500	6.4	34	47
MH	31	180.3	81.8	1200	3.4	32	45
SR	32	167.6	63.6	900	3.4	30	42
MEAN	30.2	180.3	75.7	1250	4.5	32.5	45.3
SD	2.1	6.6	7.9	225	1.4	1.5	1.9

ASTHMATIC VS. NORMAL
p value <0.001

<0.05

NS

NS

NS

NS

Age is expressed in years; HT = Height in cm; WT = weight in Kg; work load in KPM; duration = time of exercise in min; retrotracheal = position of tracheal artifact in cm from the nares; retrocardiac position of cardiac artifact in cm from the nares.

TABLE 2

INSPIRED AIR TEMPERATURE AND WATER CONTENT AND VENTILATORY AND CIRCULATORY CONSEQUENCES OF EXERCISE

ASTHMATICS

Subject	Cold				Room				Body			
	Ti	Wi	\dot{V}_E	HR	Ti	Wi	\dot{V}_E	HR	Ti	Wi	\dot{V}_E	HR
TW	-19.0	1.1	83.7	168	27.2	3.9	92.9	177	37.5	45.0	89.5	180
FF	-16.7	1.5	93.0	174	28.3	6.9	92.7	175	37.9	46.0	89.7	175
LK	-17.5	1.2	48.2	144	26.1	4.9	51.8	141	36.8	44.0	48.4	144
ML	-17.2	1.2	47.6	150	27.0	8.8	52.5	150	36.7	44.0	56.5	150
PM	-16.8	1.5	75.0	171	27.8	9.1	67.0	175	37.6	45.0	54.2	165
MS	-14.4	1.6	50.7	153	25.7	3.4	43.5	146	37.1	44.2	41.1	162
DM	-9.8	2.1	79.8	174	25.4	4.6	83.7	171	37.5	45.0	80.0	180
MR	-18.6	1.0	72.6	159	26.7	8.1	79.2	150	36.7	44.0	94.7	150
MEAN	-16.3	1.4	68.8	162	26.8	6.2	70.4	161	37.2	44.7	69.3	163
SD	3.0	0.3	17.7	12	1.0	2.3	19.4	15	0.5	0.7	21.4	14

NORMALS

ND	-17.0	1.1	132.0	144	25.7	4.9	117.0	144
CO	-16.8	1.5	82.7	162	27.5	5.2	86.4	156
NS	-18.2	1.0	95.8	168	24.4	6.7	102.0	160
SP	-18.4	1.0	123.0	180	27.4	4.5	113.0	168
MH	-17.4	1.2	87.4	160	22.7	4.7	80.9	165
SK	-18.8	1.0	60.5	162	23.8	10.8	64.2	158
MEAN	-17.8	1.1	96.9	163	25.2	6.1	93.9	159
SD	0.8	0.2	26.6	12	2.0	2.4	20.4	8

ASTHMATIC VS. NORMAL

P value	NS	NS	<0.05	NS	NS	NS	<0.05	NS
---------	----	----	-------	----	----	----	-------	----

Ti = inspired air temperature in °C; Wi = inspired water content in mg H₂O/L air; \dot{V}_E = minute ventilation in L/min (BTPS); HR = heart rate in beats/min. The headings above each of the three columns refer to the individual experiments performed with various inspired air conditions.

LEGENDS FOR FIGURES

- FIGURE 1. The effect of exercise on specific conductance (SGaw) in asthmatic and normal subjects while breathing air at subfreezing, room, and body conditions. The letters B and R below each graph represent baseline data and the response observed post-exercise respectively. The data points are mean values, and the brackets indicate one standard error of the mean. The p values below each graph were derived from a two factor analysis of variance.
- FIGURE 2. The effect of exercise on one second forced expiratory volumes (FEV₁) in asthmatic and normal subjects while breathing air at subfreezing, room and body conditions. The format is identical to Figure 1.
- FIGURE 3. The effect of exercise on residual volume (RV) in asthmatic and normal subjects while breathing air at subfreezing, room and body conditions. The format is identical to Figures 1 and 2.
- FIGURE 4. Temperature recordings from the thermocouples in the rectum and retrocardiac and retrotracheal positions in the esophagus during rest, exercise and recovery in the asthmatic subjects. The data points are mean values and the brackets represent one standard error. The left, middle and right panels represent the observations obtained while the subjects inhaled air at subfreezing, room and body temperatures respectively. R₁ represents the data obtained after one minute of breathing on the heat exchanger at rest; R_L = the data from the last minute of the rest period; Ex₁ = changes observed at the end of the first minute of exercise; Ex_L = data from the last minute of exercise; and Rec = data from the 4th minute of the recovery period.

FIGURE 5. Temperature recordings from the thermocouples in the rectum and retrocardiac and retrotracheal positions in the esophagus during rest, exercise and recovery in the normal subjects. The format is identical to that in Figure 4.

FIGURE 6. Comparison of the differences in temperature between the retrocardiac and retrotracheal thermocouples during the last minute of exercise in the asthmatic and normal subjects. The left panel displays the absolute differences observed (ΔT) while the right compares the temperature differences corrected for minute ventilation ($\Delta T/\dot{V}_E$). The first pair of bars in each graph indicates the cold experiment and the second represents the room temperature study. The heights of the bars indicate the mean and the brackets one standard error.

FIGURE 7. The relationship between the fall in retrotracheal temperature with exercise and the post-exertional percentage change in one second forced expiratory volume (FEV_1) in the asthmatic subjects. The open circles indicate the body temperature experiments. The closed circles represent room temperature data and the open squares indicate the subfreezing study.

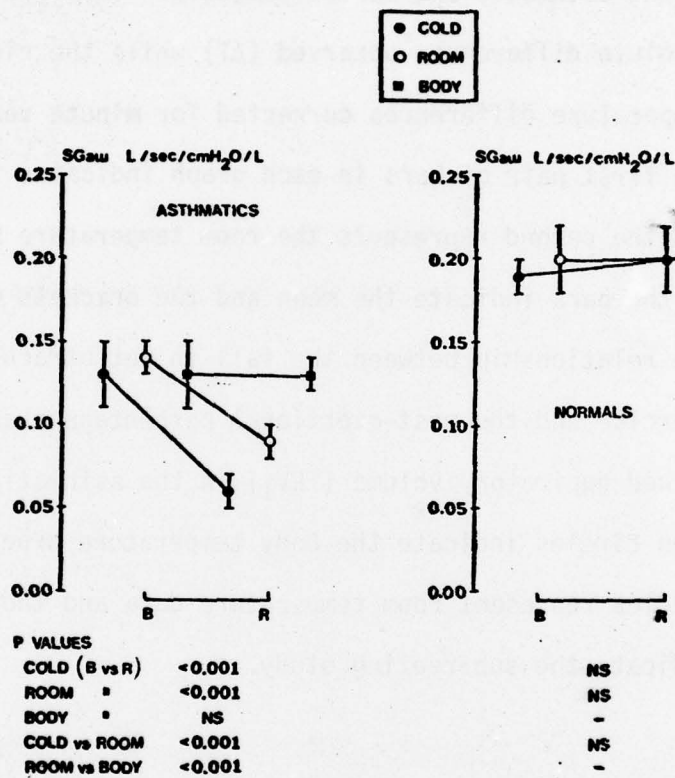


Fig. 1

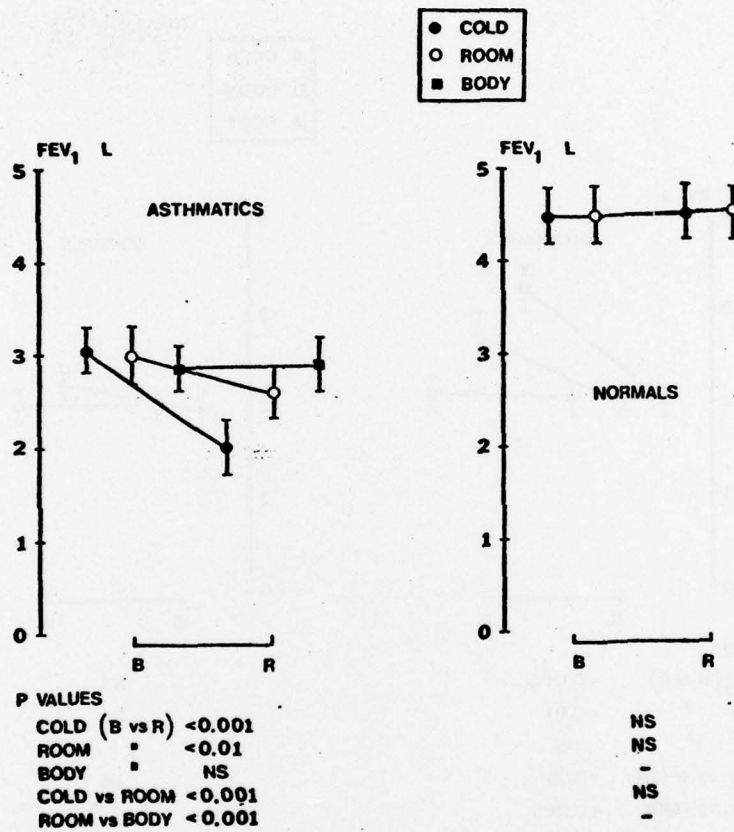


Fig. 2

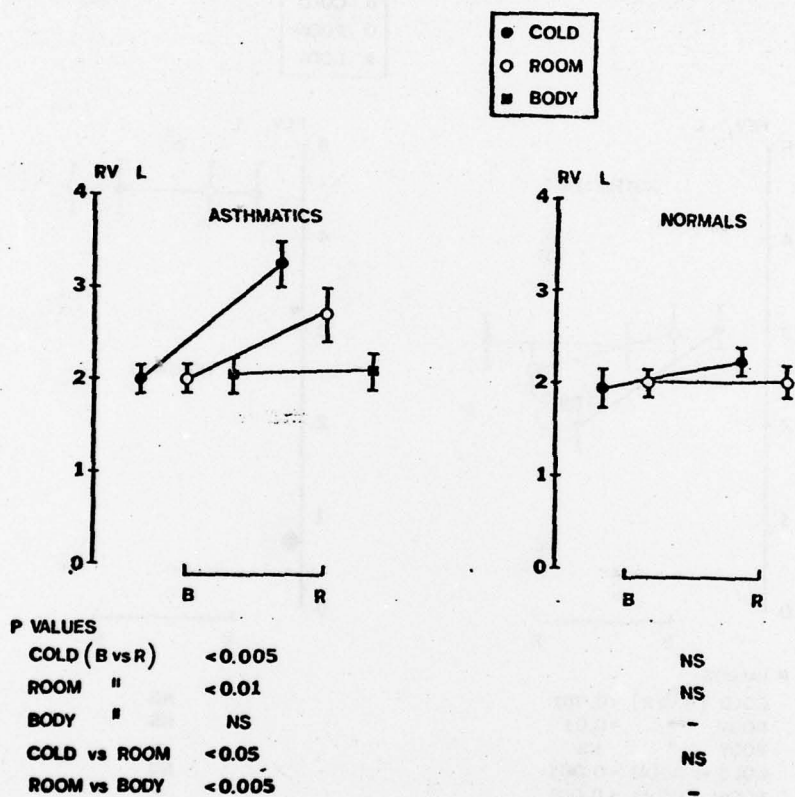


Fig. 3

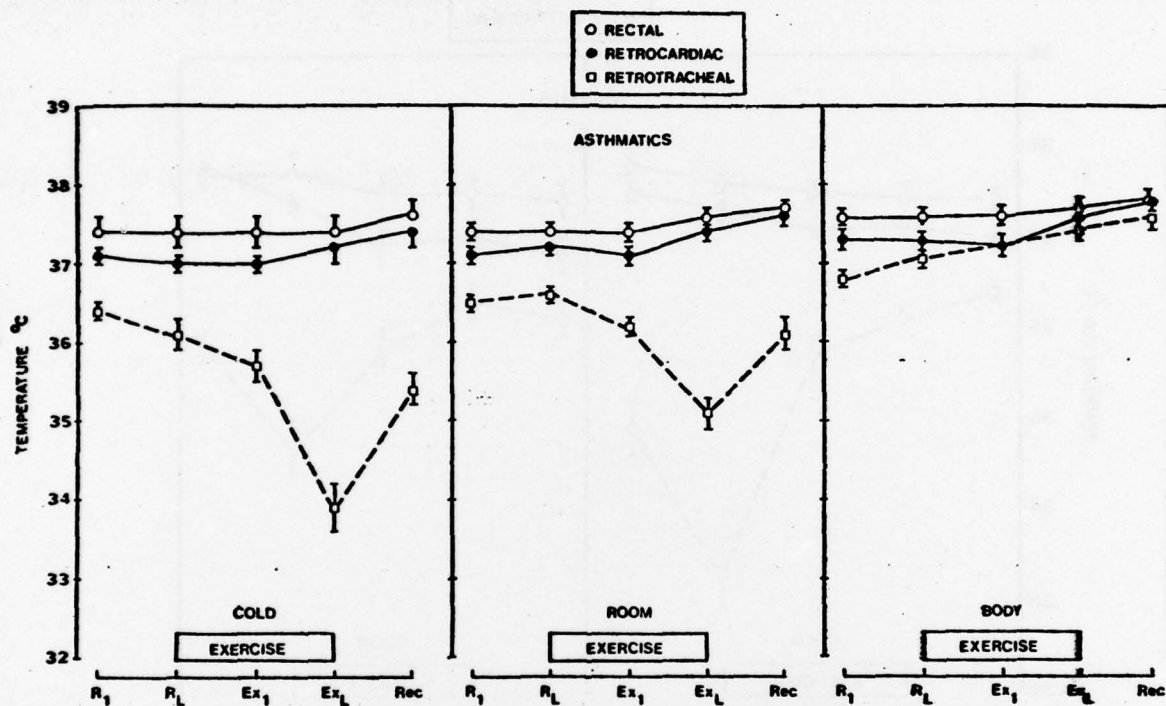


Fig. 4

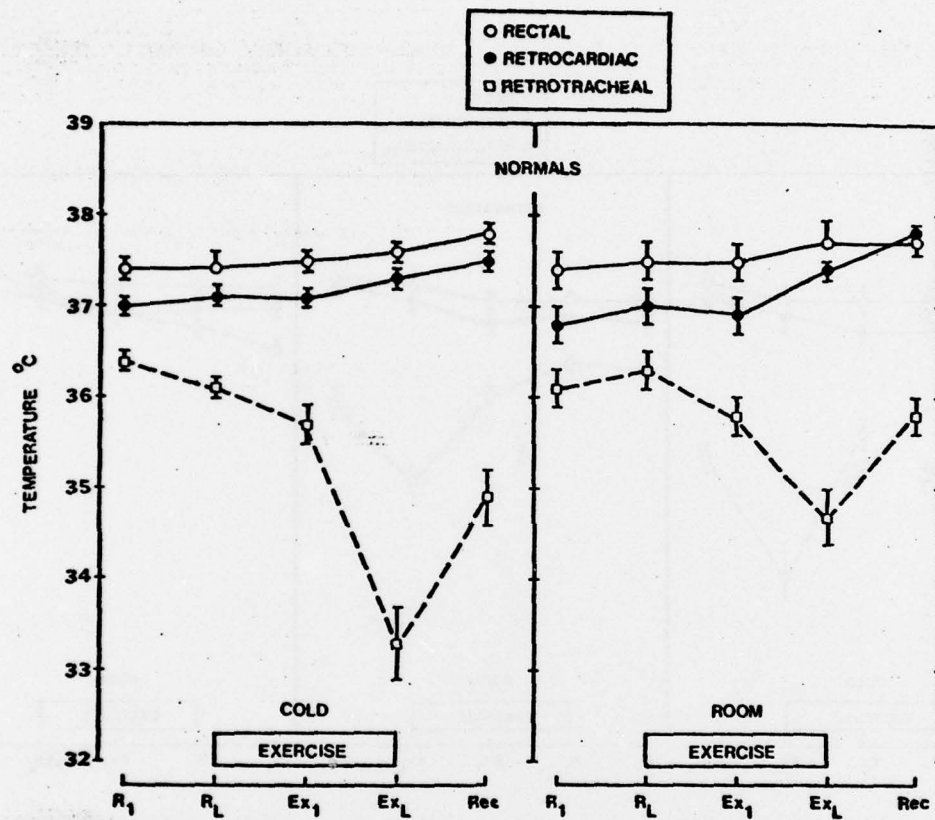


Fig. 5

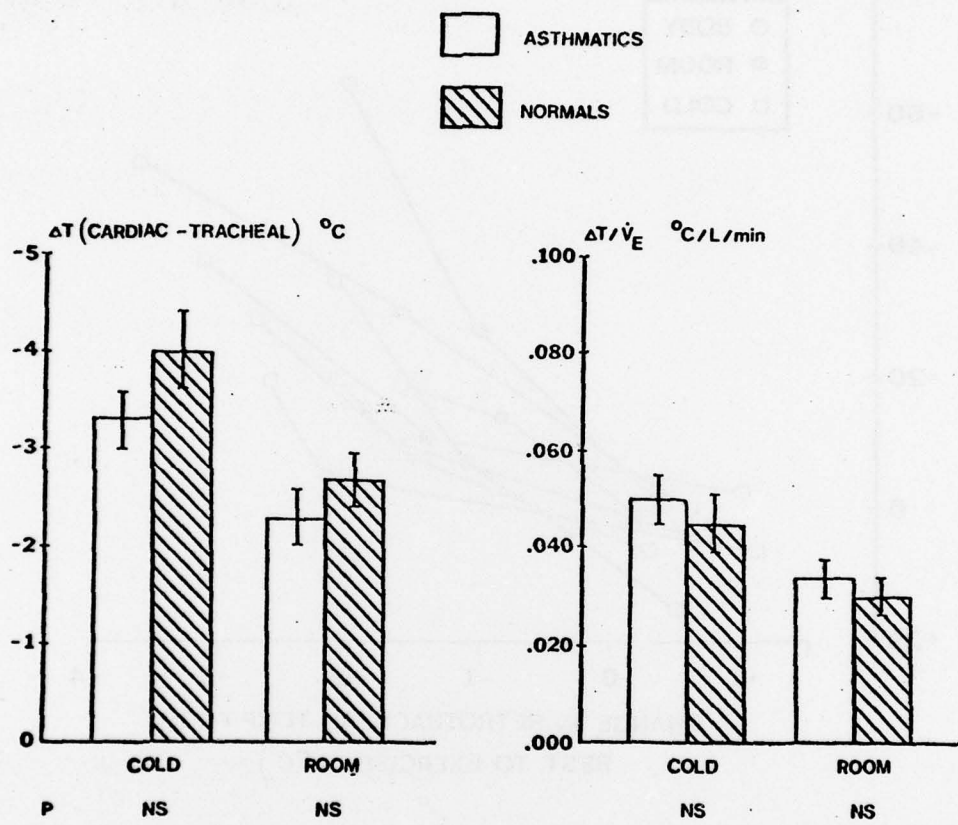


Fig. 6

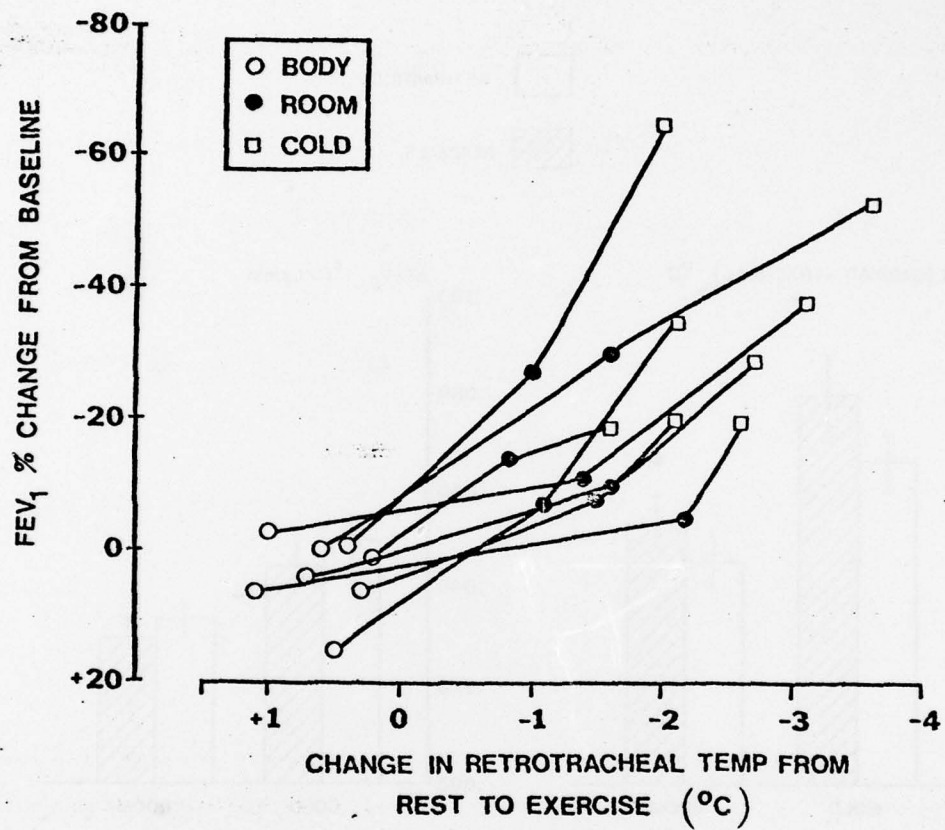


Fig. 7

1. The views, opinions, and/or findings contained in this report are those of the author(s) and should not be construed as an official Department of the Army position, policy, or decision, unless so designated by other official documentation.

2. Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on Use of Volunteers in Research.